THE RECOVERY KERNEL

Substance-Specific Navigation Series

HOW TO RECOVER FROM KRATOM

Using the Recovery Kernel Framework

Kratom · Mitragyna speciosa · Biak · Ketum · Kakuam · Thom · Herbal High · Green Maeng Da · Red Bali · White Borneo

⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE | Crisis: call 911 or text 988

KRATOM WITHDRAWAL IS OPIOID-TYPE. BUPRENORPHINE CAN MANAGE IT. ASK FOR IT.

CYP3A4 INTERACTIONS CAN DRAMATICALLY INCREASE KRATOM'S EFFECTIVE DOSE. CHECK YOUR MEDICATIONS.

A Note Before You Begin

The Recovery Kernel Compendium places kratom in the opioid withdrawal protocol without qualification: 'Opioid Withdrawal (Heroin, Fentanyl, Oxycodone, Kratom).' The opioid-type withdrawal profile is acknowledged directly in the custody medical section: 'Kratom / high-mitragynine products — MODERATE — Opioid-type withdrawal profile — Opioid-receptor dependent — Symptoms begin 12–24 hours; similar curve to opioids.'

This guide exists because the clinical system has not kept pace with kratom's rapid proliferation. An estimated 1.7 million Americans use kratom regularly, making it one of the fastest-growing substances in the US market. Yet many physicians have never heard of it, the FDA has attempted scheduling and failed, the DEA has not classified it federally, and the person experiencing kratom withdrawal in an emergency department or custody setting is frequently met with a physician who either does not recognize the clinical picture or does not know what to do about it.

Kratom is unique in this guide series for a specific reason: many people using it began by trying to solve a legitimate opioid problem. The person who switched from oxycodone to kratom to manage chronic pain without a prescription was making a pragmatic harm reduction decision. The person who used kratom to self-manage heroin withdrawal was being resourceful. Both may now have a kratom use disorder that requires the same framework as opioid use disorder — because kratom's

primary active compound, 7-hydroxymitragynine, is a partial mu-opioid receptor agonist. The solution became the new problem. The guide holds both.

Kratom also has a pharmacological identity that no other opioid in this series has: at lower doses, it is stimulant-like, not opioid-like. The dose-dependent polarity of kratom — stimulant at 2–5g, opioid at 5–15g+ — means the person using kratom for energy and focus is using a different pharmacological entity than the person using it for pain management or euphoria. Understanding which profile applies determines the recovery architecture.

Section 0 — Immediate Safety

⚠ KRATOM WITHDRAWAL IS REAL AND OPIOID-TYPE. SEVERE CASES INCLUDE INTENSE MUSCLE ACHES, VOMITING, DIARRHEA, INSOMNIA, AND ANXIETY THAT REQUIRE MEDICAL MANAGEMENT. BUPRENORPHINE HAS BEEN USED SUCCESSFULLY — ASK FOR IT BY NAME.

0.1 — High-Dose Kratom Overdose

At very high doses — particularly with concentrated kratom extracts or 7-hydroxymitragynine isolates — kratom produces opioid-type respiratory depression and sedation that can be life-threatening. Standard overdose signs apply:

- Severe sedation, difficulty waking, slowed breathing → call 911
- Naloxone: may partially reverse high-dose kratom toxicity through its opioid receptor antagonism, but naloxone's coverage of kratom's full pharmacological profile is incomplete. Administer if available and call 911.
- Mitragynine and 7-OH-mitragynine are not reliably detected on standard opioid urine screens — tell emergency personnel the specific substance.

0.2 — QTc Prolongation: The Cardiac Risk

⚠ MITRAGYNINE HAS DOCUMENTED QTc PROLONGATION POTENTIAL. COMBINED WITH OTHER QTc-PROLONGING MEDICATIONS (CERTAIN ANTIDEPRESSANTS, ANTIPSYCHOTICS, ANTIBIOTICS, ANTIFUNGALS), CARDIAC ARRHYTHMIA RISK IS ELEVATED.

Chest palpitations, racing heart, fainting, or dizziness during kratom use: cardiac evaluation (ECG) is indicated. Disclose kratom use specifically to the treating physician.

0.3 — Liver Injury Warning

⚠ KRATOM HAS BEEN DOCUMENTED TO CAUSE DRUG-INDUCED LIVER INJURY (DILI). JAUNDICE (YELLOW SKIN OR EYES), DARK URINE, SEVERE ABDOMINAL PAIN, OR EXTREME FATIGUE ARE WARNING SIGNS. STOP KRATOM AND SEEK EMERGENCY EVALUATION IMMEDIATELY.

Kratom-induced hepatotoxicity typically presents 1–8 weeks after initiating use or after dose escalation. The pattern is often cholestatic or mixed. Some cases resolve with cessation; severe cases require specialist hepatology management.

0.4 — Contamination: What May Be in the Product

- Salmonella: kratom powder has been implicated in multiple salmonella outbreaks in the US (2018 FDA advisory). Fever, diarrhea, and vomiting after kratom use may be infection rather than withdrawal or overdose.
- Heavy metals: lead, nickel, and other heavy metals have been found in kratom products above acceptable limits in testing. Chronic heavy metal exposure compounds the neurotoxic burden of kratom use.
- Alkaloid content variability: kratom powder alkaloid content varies dramatically by batch, vendor, and storage. A dose that was effective and manageable from one batch may be much higher or lower from another — dose-finding cannot be reliably transferred between products.
- Fentanyl adulteration: rare but documented. If overdose signs are more severe than expected from kratom alone: assume possible fentanyl contamination. Naloxone indicated.

Section 1 — What Kratom Did to Your Brain

THE CENTURION — The Dose-Dependent Polarity: Stimulant at Low Doses, Opioid at High Doses

Kratom (Mitragyna speciosa) contains over 40 alkaloids, but two dominate its pharmacology: mitragynine (the most abundant, ~60–66% of total alkaloids) and 7-hydroxymitragynine (7-OH-MG, present in small amounts but highly potent). Understanding these two compounds and their dose-dependent relationship explains why kratom acts as two entirely different substances depending on the dose.

1.1 — The Two Pharmacological Identities

Dose range	Primary active compound	Primary mechanism	Subjective experience
Low dose (1–5g powder)	Mitragynine (adrenergic + serotonergic effects dominate at lower concentrations)	Alpha-2 adrenergic agonism (stimulant-like); serotonin 5-HT2A agonism (mild); weak mu-opioid partial agonism	Energy, alertness, social ease, mild euphoria, reduced fatigue. Resembles stimulant more than opioid. Used by Southeast Asian workers for endurance during manual labor.
High dose (5–15g+ powder)	7-hydroxymitragynine (converted from mitragynine by CYP3A4; present in small natural amounts but disproportionately active)	Mu-opioid receptor partial agonism (primary — 7-OH-MG is 13x more potent than morphine at MOR); delta-opioid and kappa-opioid partial agonism; NMDA antagonism	Pain relief, sedation, emotional warmth, opioid-type euphoria, reduced anxiety. Resembles moderate opioid use. Used for self-managed opioid withdrawal, chronic pain, and euphoria.
Extract / 7-OH isolate (variable — equivalent to very high dose)	7-hydroxymitragynine isolate at high concentration	Predominantly mu-opioid receptor activation without the stimulant component. Approaches the pure opioid profile.	Strongest opioid-like effects; highest dependence risk; highest overdose risk; most severe withdrawal. These products are the farthest from traditional kratom use.

1.2 — The CYP3A4 Conversion: Why 7-OH-MG Is the Clinical Target

7-hydroxymitragynine is metabolically produced from mitragynine primarily by CYP3A4 in the liver. This metabolic conversion is why CYP3A4 inhibitors dramatically affect kratom's clinical effects — they cause mitragynine to accumulate while also increasing 7-OH-MG production relative to clearance. The result: at the same kratom dose, a CYP3A4 inhibitor (grapefruit, fluconazole, many HIV ARTs, some antibiotics, certain antidepressants) can produce dramatically more opioid-like effects and dramatically higher dependence risk.

1.3 — What Kratom Was Providing: Three Distinct User Profiles

User profile	What kratom was providing	Recovery architecture
Opioid replacement / self-managed withdrawal	The person who used kratom to get off prescription opioids or heroin — using kratom's opioid properties deliberately. Was solving a genuine opioid problem. Kratom worked, for a time, and then became the new problem.	Primary opioid use disorder recovery framework applies alongside kratom. Two-stage approach: stabilize the kratom dependency first, then address the underlying opioid use disorder history. The motivation for the original opioid use (pain, trauma, PTSD) must be addressed for either to stay resolved.
Chronic pain management	The person who could not access adequate prescription pain management — or who wanted to avoid the criminal justice and prescription monitoring implications of opioids — and found that high-dose kratom provided meaningful pain relief.	Pain management is the concurrent clinical target, not a secondary concern. Stopping kratom without a pain plan fails. Non-opioid pain management (buprenorphine for pain + OUD, physical therapy, interventional procedures) must be established simultaneously.
Energy, focus, and daily function	The person using low-dose kratom for the stimulant-like properties — to work longer, to manage ADHD symptoms, to maintain productivity in demanding situations. May not have recognized opioid dependency developing at escalating doses.	Energy and focus architecture from the stimulant recovery guides (dopamine support) alongside the opioid recovery framework for the physical dependence. ADHD evaluation if stimulant properties were the draw.

1.4 — The Escalation Pattern

Kratom tolerance develops through the same mu-opioid receptor downregulation mechanism as prescription opioids. The person who started at 3–5g for energy and clarity finds that the energy effect requires 6–8g, then the opioid effects emerge at that dose, and the dose that previously produced mild stimulation now produces significant opioid-like dependence. This escalation is pharmacologically identical to the shift from therapeutic to problematic opioid use — because the mechanism is the same.

The compendium's opioid escalation framework describes this exactly: 'the dose that landed in the therapeutic window initially moves toward the euphoric window as tolerance develops.' In kratom, this translates to the low-dose stimulant profile gradually shifting into the high-dose opioid profile as tolerance requires higher doses to achieve any effect.

Section 2 — Your Genomic Blueprint

Kratom's pharmacogenomics closely mirror opioid pharmacogenomics for the high-dose profile and stimulant pharmacogenomics for the low-dose profile. The defining genes are the same as for opioids — with the specific addition of CYP3A4 as the primary metabolic gene.

Gene	What it controls	Key variant	Kratom recovery relevance
CYP3A4	Primary metabolism of mitragynine → 7-hydroxymitragynine conversion AND kratom clearance	Inducers vs. inhibitors; multiple variants affecting enzyme activity	THE most clinically important pharmacogenomic factor for kratom. CYP3A4 converts mitragynine to 7-OH-MG (the primary opioid-active metabolite) AND clears mitragynine itself. CYP3A4 inhibitors (fluconazole, grapefruit, clarithromycin, many HIV ARTs, some SSRIs) dramatically increase kratom's effective opioid potency by slowing clearance and potentially increasing 7-OH-MG accumulation. Conversely, CYP3A4 inducers (rifampin, St. John's Wort, carbamazepine) accelerate clearance and can precipitate withdrawal at previously adequate doses. This is the drug-drug interaction gene for kratom.
OPRM1	Mu-opioid receptor — primary target of 7-OH-MG	A118G (rs1799971) — A/A wild type, G allele variants	The same gene that the fentanyl and opioid guides identify as primary. G allele carriers have reduced mu-opioid receptor binding affinity — they needed higher kratom doses for the same opioid-like effects and may find the opioid-like properties of kratom less reinforcing at standard doses. G allele also predicts stronger naltrexone response if extended-release naltrexone is used for kratom use disorder maintenance.
COMT	Dopamine clearance — relevant to the stimulant-like low-dose profile	Val158Met (rs4680)	Val/Val (Warrior) carriers were drawn to kratom's stimulant-like low-dose properties — the adrenergic and dopaminergic activation at lower doses fits the Warrior's need for intensity and stimulation. Recovery for this profile requires the stimulant natural architecture (exercise, competition, mastery) alongside the

			opioid endorphin rebuilding.
MTHFR	Methylation — dopamine and endorphin synthesis foundation	C677T (rs1801133), A1298C (rs1801131)	MTHFR variants reduce baseline dopamine and endorphin synthesis efficiency — potentially contributing to the vulnerability to both the stimulant and opioid effects of kratom. 5-MTHF supplementation often produces meaningful early improvement in mood and energy during recovery by addressing the synthesis deficit directly.
CYP2D6	Secondary kratom metabolism pathway	Poor metabolizer (*4/*4), intermediate, ultra-rapid	CYP2D6 plays a secondary role in mitragynine metabolism. Poor CYP2D6 metabolizers may have altered kratom pharmacokinetics — slightly longer duration from the CYP2D6 pathway contribution. More clinically significant: CYP2D6 is the primary metabolism pathway for buprenorphine (a key MAT option for kratom withdrawal) — CYP2D6 poor metabolizers may require different buprenorphine dosing.

Phenotyping Without Testing

- Did kratom produce much stronger opioid-like effects than you expected at doses others tolerated without significant effect? → Possible CYP3A4 inhibiting medication creating 7-OH-MG accumulation.
- Did kratom suddenly feel much stronger or weaker after starting a new medication? → CYP3A4 interaction. Check your medications against a drug interaction database.
- Did low-dose kratom specifically feel stimulating and clarifying before higher-dose opioid effects developed? → Warrior COMT + adrenergic low-dose kratom profile.
- Did you use kratom specifically to manage opioid withdrawal or chronic pain? → The original pain or opioid problem is a concurrent recovery target.

Section 3 — Medication-Assisted Treatment for Kratom

THE CENTURION — The Opioid Receptor Is the Target. The Opioid MAT Framework Applies.

Because kratom's high-dose dependence is mediated by mu-opioid receptor activation (through 7-hydroxymitragynine), the same MAT framework as for opioid use disorder applies. Buprenorphine has been used successfully for kratom withdrawal management in multiple case reports and series.

3.1 — Buprenorphine for Kratom Withdrawal

Evidence: Multiple case reports and clinical series describe successful buprenorphine induction for kratom withdrawal, with good symptom management and patient retention. The same partial mu-opioid agonism that makes buprenorphine effective for heroin and prescription opioid withdrawal applies to kratom.

Precipitated withdrawal caution: The same concern as with fentanyl applies here. 7-hydroxymitragynine has high mu-opioid receptor binding affinity. Buprenorphine given before adequate kratom clearance may precipitate withdrawal. The Bernese microdosing induction protocol (low-dose buprenorphine started while some kratom is still in the system, escalated gradually) has been used to avoid this. Discuss with your prescriber.

Practical access: Any DEA-registered provider can prescribe buprenorphine (since the X-waiver removal in 2023). Tell the physician specifically: 'I am dependent on kratom, which acts as a partial mu-opioid receptor agonist. Buprenorphine has been successfully used for kratom withdrawal. I am requesting evaluation for buprenorphine-assisted management.'

Duration: As with opioid use disorder, extended duration of buprenorphine treatment (months to years) is associated with better outcomes than time-limited courses. The underlying opioid receptor dependence that developed through kratom may warrant sustained MAT.

3.2 — Extended-Release Naltrexone

After complete kratom cessation (typically 7–10 days of abstinence), extended-release naltrexone (Vivitrol) blocks mu-opioid receptor activation and removes the reinforcing properties of any kratom use. OPRM1 G allele carriers show the strongest response. Requires complete clearance of kratom before initiation.

3.3 — Clonidine for Withdrawal Symptom Management

As with standard opioid withdrawal: clonidine (alpha-2 agonist) addresses the autonomic symptoms of kratom withdrawal — anxiety, sweating, restlessness, elevated heart rate and blood pressure — through the same HPA axis mechanism as in fentanyl and heroin withdrawal management. Available by prescription. Does not address the opioid-specific withdrawal symptoms as completely as buprenorphine but is often available through more settings.

3.4 — The Kratom Taper

THE CENTURION — The Self-Managed Approach — and Its Limitations

Kratom's opioid-type dependence is theoretically amenable to tapering, and many people attempt self-managed kratom tapers before seeking clinical support. The principle is sound — gradual dose reduction allows receptor adaptation — but the execution faces specific challenges:

- Alkaloid variability between products: a taper based on grams of powder cannot be precisely executed because alkaloid content varies dramatically between batches and vendors. Switching vendors mid-taper introduces uncontrolled variables.
- Dose measurement precision: kratom is typically measured by gram weight, not by alkaloid content. True precision tapering requires consistent product from a consistent source.
- The extract trap: high-extract products are far more difficult to taper than powder because the alkaloid concentration variability is even higher.
- A general taper framework (not medical advice — discuss with a physician): reduce total daily dose by 10% every 1–2 weeks. Do not jump more than 10% per reduction. Hold at each step until stable before reducing further. At low daily doses (1–2g total), cessation may be more manageable than further tapering.

Section 4 — Biological Reconstruction

Kratom biological reconstruction combines the opioid recovery framework (endorphin system rebuilding, MOR receptor recovery) with elements of the stimulant recovery framework (dopamine reconstruction for the adrenergic low-dose component) plus kratom-specific hepatic support.

4.1 — The Methylation Foundation

Supplement	Dose	Timing	Kratom-specific notes
5-MTHF (L-methylfolate)	400–1000mcg	Morning	Endorphin and dopamine synthesis support — covers both the opioid and stimulant recovery dimensions of kratom. For MTHFR variant carriers whose energy or mood deficit drove kratom use, 5-MTHF often produces meaningful early improvement.
Methylcobalamin	1000–2000mcg sublingual	Morning	Methylation support; neurological recovery support. Sublingual form for reliable absorption. Active form — not cyanocobalamin.
P5P + Magnesium glycinate	P5P 25–50mg; Mg 400–600mg	Morning; Mg evening	Cofactor stack for neurotransmitter synthesis and NMDA modulation during withdrawal. Kratom's NMDA antagonism component means magnesium's NMDA-buffering role is relevant during withdrawal as with ketamine and DXM recovery.

4.2 — Endorphin System Reconstruction

- DLPA (DL-Phenylalanine, 750–1500mg morning): enkephalinase inhibitor — extends the life of endogenous opioids. Primary opioid recovery supplement applied directly to kratom recovery. Supports the endogenous opioid system that kratom was activating pharmacologically.
- NAC (600–1800mg daily, divided): glutamate homeostasis; craving reduction. Multiple trials supporting NAC for opioid-spectrum craving reduction.

- Omega-3 DHA + EPA (2–4g daily): MOR receptor membrane environment; anti-neuroinflammatory. Same rationale as fentanyl and opioid recovery.
- Vitamin D3 (3000–5000 IU with K2, morning with fat): endorphin synthesis support; documented deficiency in opioid use disorder populations.

4.3 — Dopamine Reconstruction (for stimulant-profile users)

- L-Tyrosine (500–1000mg, morning, away from protein): dopamine precursor for the adrenergic component of kratom's low-dose effects.
- Rhodiola rosea (300mg, morning): dopamine and serotonin modulation; adaptogenic anti-fatigue. The energy deficit of kratom cessation in low-dose users is partly dopaminergic.
- Brain Stack — Uridine + DHA + CDP-Choline: D1/D2 receptor density restoration for users whose primary kratom use was at the dopaminergic/adrenergic low-dose range.

4.4 — Liver Support — Kratom-Specific Priority

⚠ LIVER FUNCTION TESTING (AST, ALT, GGT, BILIRUBIN) IS RECOMMENDED FOR ALL SIGNIFICANT KRATOM USE HISTORIES. KRATOM-INDUCED HEPATOTOXICITY IS DOCUMENTED AND MAY REQUIRE HEPATOLOGY MANAGEMENT.

- NAC (serving double duty here — liver support + craving reduction): glutathione precursor directly relevant for hepatic recovery from kratom-induced liver injury.
- Milk thistle (silymarin, 150–300mg standardized extract, daily): hepatoprotective; antioxidant; specifically relevant for kratom liver injury recovery.
- Alpha-lipoic acid (300mg, morning, away from meals): mitochondrial antioxidant; hepatoprotective; blood-brain barrier crossing.
- Avoid alcohol during recovery: kratom-induced hepatotoxicity combined with alcohol creates compound hepatic stress. Complete alcohol cessation while liver enzymes are elevated.

4.5 — The Withdrawal Timeline

Timeline	Biology	Experience	What to do

Hours 12–24 (onset)	MOR receptor upregulation beginning; endogenous opioid system attempting to compensate for withdrawal of kratom agonism; HPA axis activation	Compendium: 'Endorphin system offline → HPA axis in overdrive.' Restlessness, anxiety, yawning, runny nose, mild muscle aches, craving beginning	Begin supplement stack. Clonidine if prescribed. First buprenorphine dose if initiating MAT with physician. The 60–75 BPM, warm-frequency sonic protocol from the compendium begins here.
Hours 24–72 (peak)	Peak MOR upregulation; endorphin system at maximum deficit; HPA axis at maximum activation; sleep architecture severely disrupted	Moderate opioid-type withdrawal: body aches, insomnia, anxiety, restlessness, GI symptoms (nausea, diarrhea), depression, intense craving. Generally less severe than heroin withdrawal; more severe than typical prescription opioid withdrawal.	MAT if available. Clonidine for autonomic symptoms. Loperamide for GI symptoms (at directed doses). Magnesium for muscle cramping. DLPA and NAC for craving substrate. Rest priority.
Days 3–7	Acute withdrawal subsiding; MOR receptor sensitivity beginning to recover; sleep slowly improving	Improvement with variability; depression often prominent as acute withdrawal resolves; motivation and energy below baseline; craving less acute but more emotionally driven	Full supplement stack active. Exercise beginning even if brief. 5-HTP (50mg evening) for mood and sleep. The opioid withdrawal sonic protocol from compendium: PAWS phase — 'singer-songwriter, folk, acoustic, human connection and warmth.'
Weeks 2–12 (PAWS)	Post-Acute Withdrawal Syndrome: episodic endorphin system deficits, dysphoria, anxiety, sleep disruption, and craving — reflecting the slower recovery of MOR receptor density and endogenous opioid production	Episodic nature of PAWS is characteristic and often misunderstood as relapse risk. Good days followed by bad days without apparent trigger. Depression that lifts and returns. Craving that seems to diminish then surges.	PAWS documentation: track each episode with date, duration, severity, trigger if any. Three weeks of documentation reveals the pattern. The episodic nature is normal and temporary. NAC, DLPA, exercise, and connection remain the primary management.

4.6 — Sleep Protocol

Kratom withdrawal produces severe insomnia — the opioid system's role in sleep architecture means its sudden absence disrupts all sleep stages. The compendium's

opioid withdrawal sonic protocol applies directly: '60–75 BPM, warm frequencies (bass, mid-range), 528 Hz, ambient, new age, classical strings, nature sounds, legato, no silence gaps.'

- Magnesium glycinate: 400mg elemental evening — GABA support and anxiety reduction
- Melatonin: 0.5–1mg sublingual, consistent bedtime — circadian signal
- Glycine: 3–5g, 30 min before bed — temperature reduction and NMDA co-agonism
- L-Theanine: 200mg evening — alpha-wave promotion without sedation
- Hydroxyzine (prescription): non-addictive antihistamine-anxiolytic; useful during acute withdrawal insomnia

4.7 — Peptide Support

- BPC-157 (daily, oral): mu-opioid receptor pathway modulation; gut-brain axis repair from kratom's GI effects; dopamine and serotonin normalization.
- Selank (Weeks 1–8 as needed): GABA and serotonin modulation; non-addictive anxiolytic; addresses the anxiety of opioid system withdrawal without addiction risk.
- Semax (Week 2 onwards): BDNF upregulation; cognitive repair; prefrontal cortex support for the impulse control and decision-making impaired by chronic mu-opioid receptor activation.

Section 5 — The Kratom Craving — Understanding and Interrupting

THE CENTURION — The Dual Craving: Opioid-Type Relief Seeking and Stimulant-Type Energy Seeking

Kratom cravings have two faces depending on which profile was primary. For the high-dose opioid-profile user: the craving is for pain relief, emotional warmth, and the absence of withdrawal. For the low-dose stimulant-profile user: the craving is for energy, focus, and productivity. Both are real. Both have specific triggers. Both require specific interrupts.

Trigger	The underlying state	The interrupt
Physical pain	The opioid profile craving most commonly fires when pain is present — the kratom was genuinely managing pain. Stopping without a pain plan means the pain triggers a direct, pharmacologically rational craving.	Pain management plan execution (Section 3 — buprenorphine for pain + OUD, physical therapy, NSAIDs, non-opioid interventions). The craving is pointing at real pain. Address the pain, not just the craving.
Fatigue / low energy / morning demand	The low-dose stimulant profile craving fires when energy is needed — a work deadline, physical demand, the morning when alertness is required. Kratom was the reliable energy source.	Vigorous exercise (20–30 min, even brief) produces the fastest natural dopamine and norepinephrine activation. Cold shower. Caffeine (managed — not a new dependency). The supplement stack is not an acute energy substitute; exercise is.
Kratom product visible or accessible	Environmental cue conditioning — the sight, smell, or proximity of kratom powder fires the anticipatory craving circuit regardless of conscious intention.	Remove all kratom products from the home, car, and workspace completely. All of it. The environmental removal is more reliable than resistance. Dispose of it — do not 'keep it just in case.'
Opioid withdrawal symptoms (PAWS)	The episodic dysphoria, body aches, and restlessness of PAWS fire the opioid-profile craving through the same mechanism as the fentanyl guide: 'my body feels like withdrawal, therefore the solution is the substance that relieves withdrawal.'	Name PAWS: 'This is a PAWS episode, not withdrawal from current cessation. I have not used. This will pass.' DLPA and NAC for the endorphin substrate of the craving. Contact your support structure. The PAWS documentation you have been keeping reveals this is a pattern with a recovery trajectory.

THE KRATOM CRAVING INTERRUPT — 5-MINUTE PROTOCOL

1. IDENTIFY THE CRAVING TYPE. Is this opioid-profile (pain, withdrawal, warmth-seeking) or stimulant-profile (energy, focus, productivity-seeking)? The specific underlying state determines the specific additional response.
2. TAKE DLPA AND NAC. The endogenous opioid and glutamate substrate of the craving. These are pharmacological interventions for a pharmacological state.
3. EXECUTE 4-7-8 BREATHING — SIX CYCLES. Parasympathetic activation for the HPA axis overdrive of opioid withdrawal craving.
4. FOR PAIN-DRIVEN CRAVING: execute the pain management plan. Contact your prescriber if pain is above threshold. The craving is accurate — the pain is real. The solution must be real too.
5. FOR ENERGY/FOCUS CRAVING: vigorous movement for 10+ minutes immediately. The adrenergic activation of exercise provides a genuine, albeit brief, alternative to kratom's adrenergic effects at low doses.
6. CONTACT YOUR SUPPORT STRUCTURE. Name the trigger. Name the type. The isolation that kratom use often involves is itself a craving driver — connection is the counter-substance.
7. DOCUMENT. Trigger, type, intensity, action, outcome. Three weeks of documentation reveals the specific PAWS pattern and the triggers most associated with relapse risk for this specific person.

Section 6 — Building the Natural Architecture for What Kratom Provided

THE CENTURION — C-28 Applied to Kratom — Two Natural Architectures for Two Pharmacological Profiles

Because kratom has two distinct functional profiles depending on dose, the natural porting architecture must address both:

For the Opioid-Profile User: The Endorphin Architecture

From the fentanyl guide's natural porting framework — applied identically here because the receptor target is the same:

- Vigorous sustained exercise (45+ min daily): the primary endorphin activator. Direct MOR activation through endogenous opioid release. Non-optional.
- Genuine human connection and physical safe touch: oxytocin and endorphin co-release through the social bonding pathway.
- Cold exposure: endorphin surge and norepinephrine activation simultaneously.
- Service to others with chronic pain or opioid history: the specific meaning and MOR activation of genuine contribution.
- Low-dose naltrexone (LDN, 1.5–4.5mg nightly by prescription): paradoxically upregulates endorphin production through brief receptor blockade. Particularly relevant if chronic pain drove kratom use — LDN has evidence for fibromyalgia and chronic pain conditions.

For the Stimulant-Profile User: The Dopamine-Adrenergic Architecture

From the stimulant recovery guides — applied to the adrenergic/dopaminergic low-dose kratom profile:

- High-intensity exercise with competitive element: the natural dopamine-norepinephrine activation that matches the stimulant profile's low-dose kratom effect at the biological level.
- Mastery at the skill frontier: flow state dopamine through the challenge-skill edge.
- ADHD treatment if indicated: the Warrior-COMT ADHD profile that drove low-dose stimulant-seeking kratom use. Treatment removes the primary driver.

- Caffeine strategically (if managed): not a substitute for kratom but a genuine adrenergic tool for the energy profile. The energy drink intelligence guide's strategic caffeine architecture applies.

Pain Management: The Third Natural Architecture

For users whose kratom use was primarily pain management, the natural porting must include a genuine pain management plan. This is not optional:

- Physical therapy: the most evidence-based non-pharmacological pain intervention for most musculoskeletal pain.
- Exercise-induced endorphin analgesia: directly reduces pain perception through the same MOR pathway kratom was activating — at lower intensity but sustainably.
- Buprenorphine for pain + OUD: addresses both pain and opioid receptor dependency simultaneously.
- Non-pharmacological pain interventions: mindfulness-based pain reduction (MBSR), acupuncture, TENS, heat/cold therapy, sleep optimization for pain management.

Section 7 — Navigating the Recovery Kernel: Your Protocol Map

Phase	Protocols	Primary focus	Kratom-specific notes
Medical Safety Assessment (Days 0–7)	Physician; liver function testing; ECG; buprenorphine evaluation	Liver enzymes; QTc; buprenorphine induction decision; clonidine for withdrawal	Liver function testing is kratom-specific and urgent — hepatotoxicity can develop rapidly. ECG for QTc with kratom use history. The buprenorphine conversation: bring this guide's framing to the physician encounter.
Withdrawal Management (Days 1–7)	MAT (buprenorphine preferred); clonidine; loperamide; sleep stack	Acute opioid-type withdrawal management; HPA axis support; sleep	The opioid withdrawal sonic protocol from compendium begins Day 1. Supplement stack Day 1. MAT initiation if proceeding with buprenorphine — discuss induction timing with prescriber to avoid precipitated withdrawal.
Biological Reconstruction (Weeks 1–8)	DLPA, NAC, 5-MTHF, BDNF building, endorphin architecture	Endorphin system rebuilding; dopamine recovery (if stimulant profile); liver recovery; sleep normalization	DLPA and NAC from Day 1. 5-MTHF from Day 1. Liver support stack concurrent. Exercise starting Week 1 even if brief. The endorphin and dopamine architectures mirror the fentanyl and cocaine guides respectively — apply the appropriate one based on primary kratom profile.
Pain Management Track (Weeks 1–12+, ongoing)	Pain management physician; physical therapist; buprenorphine if pain + OUD	Non-opioid chronic pain management; physical therapy; buprenorphine optimization for pain	The person using kratom for chronic pain has not had the pain addressed — they have managed it with kratom. Stopping without a concurrent pain plan will fail. This track runs alongside recovery indefinitely.
Long Game (Months 3+)	Natural endorphin and dopamine architectures; pain management sustainability; service	Genuine endorphin system operating; pain managed without opioid-class substances; the life beyond kratom management	The person who has rebuilt the endorphin architecture through exercise, connection, and meaning — AND who has a genuine pain management plan — has removed both the biological and functional drivers of kratom return. This is the sustainable end state.

Section 8 — The Medical Context: The Regulatory Limbo

Kratom's legal and regulatory status creates specific challenges that no other substance in this guide series faces:

- Federally legal in the US (as of 2026): the DEA backed off scheduling after massive public opposition in 2016. Kratom is not a controlled substance federally, but is banned in Alabama, Arkansas, Indiana, Rhode Island, Vermont, and Wisconsin, and several municipalities.
- No FDA-approved use: kratom is not FDA-approved for any medical purpose. The FDA has issued multiple warnings about kratom products and has attempted to remove them from the market through import alerts and enforcement actions.
- The Kratom Consumer Protection Act (KCPA): model legislation developed by the American Kratom Association that would regulate kratom for quality, labeling, and age restrictions without banning it. Has been passed in several states. Provides a regulatory framework for harm reduction within legal use.
- Clinical documentation challenge: because kratom is not in standard drug panels and is not a controlled substance, the medical record may not accurately reflect kratom use history. Specifically disclose to all treating physicians.
- The Having the Physician Conversation: 'I have a kratom use disorder characterized by dependence on mitragynine and 7-hydroxymitragynine — partial mu-opioid receptor agonists. I am experiencing opioid-type withdrawal. I have been researching buprenorphine-assisted management for kratom withdrawal and I am requesting evaluation for this approach.' This framing requires the physician to engage with the clinical picture, not dismiss it.

A Final Word

THE CENTURION — The Plant That Was Supposed to Be the Solution — And Then Became the Problem

Many people reading this guide came to kratom with the best possible reasons. They were getting off opioids without a prescriber. They were managing pain that the medical system had either undertreated or overtreated. They were finding energy for a demanding life without the criminal implications of stimulants. They were doing harm reduction, imperfectly, with what was available.

The compendium's C-37 principle — every substance preference is a signal — applies here with specific compassion. The kratom preference was a signal: a pain system that needed management, an energy system that needed support, an opioid-receptor-mediated relief from something that genuinely hurt. The signal was accurate. The solution was temporary and developed its own problem.

Recovery from kratom is not a moral correction. It is finding what the plant was providing — pain relief, energy, endorphin-mediated warmth — through means that do not themselves require management. The endorphin system the body was given can be rebuilt to provide the pain buffer that kratom was pharmacologically supplying. The dopamine architecture can provide the energy and focus that low-dose kratom was adrenergically delivering.

The opioid MAT framework that the clinical system largely reserved for heroin and fentanyl users applies to you. Buprenorphine works for kratom withdrawal. Ask for it by name. The physician who does not know about kratom's mu-opioid mechanism needs to be informed — and this guide provides the language to do that.

You were solving a real problem. Recovery is solving it better.

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American Kratom Association: americankratom.org | Treatment locator: findtreatment.gov | Crisis: 988

Refer to the Recovery Kernel Complete Centurion Compendium for all referenced protocols.

Crisis: call or text 988 | Emergency: 911 | SAMHSA: 1-800-662-4357